REMARKS

This is in response to the United States Patent and Trademark Office communication dated May 19, 2004, to comply the claims in the response to the United States Patent and Trademark Office Action filed February 26, 2004. No new matter has been added. The following Remarks are as previously presented.

Reconsideration of the above-identified application in view of the amendments above and the remarks following is respectfully requested.

Claims 23, 24, 27-36, 38 and 39 are in this case. Claims 23, 24, 30-33, 36, 38 and 39 have been rejected. Claims 27-29, 34 and 35 have been objected to. Claims 36, 38 and 39 have now been canceled. Claims 23, 27 and 30 have now been amended.

Applicant acknowledges with appreciation the indication of allowable subject matter of claims 27, 28, 29, 34 and 35.

35 U.S.C. § 112, Second Paragraph, Rejections

The Examiner has rejected claim 30 under 35 U.S.C. § 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which Applicant regards as the invention. The Examiners rejections are respectfully traversed. Claim 30 has now been amended.

Specifically, the Examiner points out that claim 30 is indefinite in its recitation of "said exogenous factor" because the phrase lacks antecedent basis.

Claim 30 has now been amended to include the phrase "an exogenous factor", to thereby overcome the Examiner's rejection with respect to this claim.

35 U.S.C. § 102(b) Rejections

The Examiner has rejected claims 23, 24, 31-33, 36, 38 and 39 under 35 U.S.C. 102(b) as being anticipated by Koh et al. (1995). Claims 36, 38 and 39 have now been cancelled. Claims 23 and 27 have now been amended.

The Examiner states that Koh et al. disclose successful engraftment of fetal canine cadriomyocytes into the hearts of adult CXMD dogs. In Koh's study, confocal laser scanning microscopy revealed the presence of connexin43, the major

constituent of the cardiac gap junction, at the donor-host cardiomyocyte border. Thus, the Examiner concludes that the present invention is disclosed by the prior art.

The present invention relates to the transplantation of cellular grafts which express exogenous ion channels to modify the electrophysiological function of any excitable tissue region transplanted therewith.

The present inventors were the first to show that introduction of cells which express exogenous ion channels into an excitable tissue can be used to control the electrophysiological function of excitable tissues, to thereby treat various disorders associated with such tissues (see Example 5 of the Examples section of the instant application as well as the Appendix section, which was attached to the response filed March 3, 2003).

Although, Koh and co-workers disclosed the transplantation of genetically modified canine cadriomyocytes into the heart of a dog, these cardiomyocytes were genetically modified to express the genetic marker β-Gal which was merely used as a transgene reporter for monitoring cell fate and not exogenous ion channel polynucleotides. The objective of this methodology was to increase the number of functional cardiomyocytes in a diseased heart (i.e., increasing the cell mass) and not to manipulate their physiological properties.

Koh et al. did not describe nor did they suggest the use of cells transfected with ion channel coding sequences for the purpose of modifying the electrophysiological function of excitable tissues.

Applicant would like to emphasize that it is the exogenous ion channels and not the gap junction forming protein that is essential to manipulate the physiological properties of an excitable tissue.

Notwithstanding the above and in the interest of expediting prosecution of this case, Applicant has elected to add limitations to claims 23 and 36 to distinguish the methods of the present invention from the prior art.

Thus, claim 23 has been amended to recite the following:

"A method of modifying the electrophysiological function of an excitable tissue region of an individual, the method comprising:

(a) providing cells expressing <u>an exogenous polypeptide</u> forming a functional ion channel or transporter; and

(b) implanting said cells into the excitable tissue region, such that each implanted cell forms:

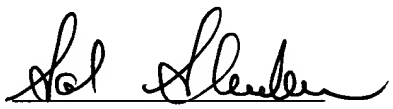
(i) gap junctions with at least one cell of the excitable tissue region; and

(ii) a functional ion channel or transporter;

thereby modifying the electrophysiological function of the excitable tissue region." (Emphasis added)

In view of the above amendments and remarks it is respectfully submitted that claims 23, 24 and 27-35 are now in condition for allowance. Prompt Notice of Allowance is respectfully and earnestly solicited.

Respectfully submitted,



Sol Sheinbein
Registration No. 25,457

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